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Public Health Implications of 1990 Air Toxics Concentrations across the United States

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Occupational and toxicological studies have demonstrated adverse health effects from exposure to toxic air contaminants. Data on outdoor levels of toxic air contaminants have not been available for most communities in the United States, making it difficult to assess the potential for adverse human health effects from general population exposures. Emissions data from stationary and mobile sources are used in an atmospheric dispersion model to estimate outdoor concentrations of 148 toxic air contaminants for each of the 60,803 census tracts in the contiguous United States for 1990. Outdoor concentrations of air toxics were compared to previously defined benchmark concentrations for cancer and noncancer health effects. Benchmark concentrations are based on standard toxicological references and represent air toxic levels above which health risks may occur. The number of benchmark concentrations exceeded by modeled concentrations ranged from 8 to 32 per census tract, with a mean of 14. Estimated concentrations of benzene, formaldehyde, and 1,3-butadiene were greater than cancer benchmark concentrations in over 90% of the census tracts. Approximately 10% of all census tracts had estimated concentrations of one or more carcinogenic HAPs greater than a 1-in-10,000 risk level. Twenty-two pollutants with chronic toxicity benchmark concentrations had modeled concentrations in excess of these benchmarks, and approximately 200 census tracts had a modeled concentration 100 times the benchmark for at least one of these pollutants. This comprehensive assessment of air toxics concentrations across the United States indicates hazardous air pollutants may pose a potential public health problem. *Key words*: air toxics, atmospheric dispersion models, exposure assessment, risk assessment. *Environ Health Perspect* 106:245–251 (1998). [Online 6 April 1998]
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There has been public concern regarding the health effects of air pollution on health for the past 50 years, much of it prompted by the dramatic pollution episodes in Donora, Pennsylvania, and London, England (*1,2*). However, much of the attention resulting from these episodes has focused on those pollutants designated as criteria pollutants in the Clean Air Act, such as particulate matter, ozone, and lead. Relatively little is known about the potential health effects of other toxic air pollutants, a number of which are designated as hazardous air pollutants (HAPs) in the Clean Air Act. HAPs, also known as air toxics, have been associated with a variety of adverse health outcomes, including cancer and noncancer effects such as neurological, reproductive, and developmental effects, mostly through occupational and animal studies (*3*).

There have been some previous efforts to characterize the potential impacts of hazardous air pollutants (*4–9*). Some studies have attempted to assess differential impacts of air toxics on communities of color using emissions estimates, mostly from the Toxics Release Inventory (TRI), which contains emissions estimates from major manufacturers in the United States (*7,9*). Other analyses have attempted to characterize the potential public health impacts of air toxics (*4–6,8,9*). One set of studies uses monitoring data and concentrations estimated by dispersion modeling of emissions from a subset of commercial and industrial facilities to evaluate potential noncancer health effects (*4–6*). These studies found that outdoor concentrations were often greater than benchmarks representing thresholds for potential public health impacts. Another study using monitoring and modeling data found that air toxics posed a potential cancer risk (*8*). These analyses represent important steps in assessing the public health implications of air toxics. However, they rely on emissions data for a limited selection of emissions sources and monitoring data for a limited selection of pollutants and locations to assess broad public health impacts and typical population exposures. One of the important limitations in our ability to better understand the potential health effects of HAPs is a lack of outdoor concentration data with broad geographic coverage and broad coverage of emissions sources. While some monitoring data are available, they are limited in terms of the number of pollutants monitored and consistency of geographic coverage (*10*).

Given the paucity of measured data, an alternative approach to assessing outdoor concentrations of HAPs is to use atmospheric dispersion models. Previously, existing analytical tools and data have been used to model air toxic concentrations for small geographic areas or for a limited number of pollutants (*4,6,7*). These methods can be applied with a broader, national geographic scope to evaluate the dispersion of multiple air toxics from multiple sources. A recent analysis, conducted as part of the EPA Cumulative Exposure Project, has modeled outdoor concentrations of air toxics across the contiguous United States (*11*) to help address the lack of data on outdoor concentrations. Emissions data from stationary and mobile sources are used as inputs into a dispersion model that estimates 1990 average outdoor concentrations of 148 air toxics for every census tract in the contiguous United States.

This comprehensive evaluation of the potential public health implications of outdoor air toxics concentrations across the United States assesses whether modeled concentrations are above or below a level that may warrant concern. Estimated outdoor concentrations are used as a reasonable proxy for potential exposure in making relative comparisons of hazard and performing screening level analysis. Outdoor concentrations of air toxics are compared to previously defined levels (benchmark concentrations), that represent thresholds of concern for potential adverse public health impacts (*12*).

Methods

Outdoor concentrations of air toxics. Outdoor concentrations of HAPs were estimated using a Gaussian dispersion model (*11,13*). The Assessment System for Population Exposure Nationwide (ASPEN)

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The views expressed in this report are those of the authors. They do not necessarily represent those of the U.S. Environmental Protection Agency.

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used in this study is a modified version of the EPA Human Exposure Model [HEM; (*13*)], a standard tool designed to model long-term concentrations over large spatial scales. Long-term average concentrations of HAPs were calculated at the census tract level based on emissions rates of the HAPs and frequencies of various meteorological conditions, including wind speed, wind direction, and atmospheric stability. In addition, the model used in this analysis incorporates simplified treatment of atmospheric processes such as decay, secondary formation, and deposition. There are 60,803 census tracts in the contiguous United States that vary in physical size but have typically 4,000–5,000 residents.

The pollutants chosen for modeling were based on the list of 189 HAPs in section 112 of the 1990 Clean Air Act Amendments. A baseline year of 1990 was selected for modeling. Available emissions data were reviewed, and appropriate data were identified for 148 HAPs.

A national inventory of HAP emissions was developed for this study as a required input to the dispersion model. For large manufacturing sources, emissions data contained in the EPA's TRI were used (*14*). Emissions estimates were developed for other sources, such as large combustion sources, automobiles, and dry cleaners, using HAP speciation data in combination with the EPA's extensive national inventories of 1990 emissions of total volatile organic compounds (VOCs) and particulate matter (PM) (*15,16*). HAP emissions were derived from VOC and PM emissions estimates by applying industry-specific and process-specific estimates of the presence of particular HAPs in a VOC or PM emissions stream (*11*). Details are described by Rosenbaum et al. (*11*). Alaska and Hawaii are not included in this study because the

Table 1. Classification of hazardous air pollutant (HAP) health effects information for comparison with estimated outdoor concentrations

Health effect	Tier[a]	HAPs with value (*n*)	Health effect value
Cancer[b]	I	40	EPA inhalation unit risk for carcinogenicity
	II	37	EPA oral unit risk for carcinogenicity expressed in inhalation units; California EPA inhalation unit risk estimate
Chronic noncancer[c]	I	33	EPA inhalation reference concentration
	II	57	EPA provisional inhalation reference concentrations; California EPA reference exposure level; Agency for Toxic Substances and Disease Registry minimum risk levels
Acute[d]	II	16	EPA inhalation reference concentration (developmental), EPA LOC/1,000

LOC, levels of concern. Development of toxicity data has previously been described by Caldwell et al. (*12*).

[a]The tiers indicate the level of priority for use of toxicological data and uncertainty in the data. Tier I information is highest priority data with most confidence.

[b]The pollutant groups arsenic, beryllium, cadmium, chromium, lead, and nickel compounds have each been assigned a single Tier I cancer benchmark concentration. Other HAPs with Tier I cancer benchmarks are individual pollutants.

[c]The pollutant groups manganese, cadmium, and selenium compounds have each been assigned a single Tier II chronic benchmark concentration. Other HAPs with Tier II chronic benchmarks are individual pollutants.

[d]The pollutant group chromium compounds has been assigned a single Tier II acute benchmark concentration. Other HAPs with Tier II acute benchmarks are individual pollutants. There is one Tier I acute benchmark concentration.

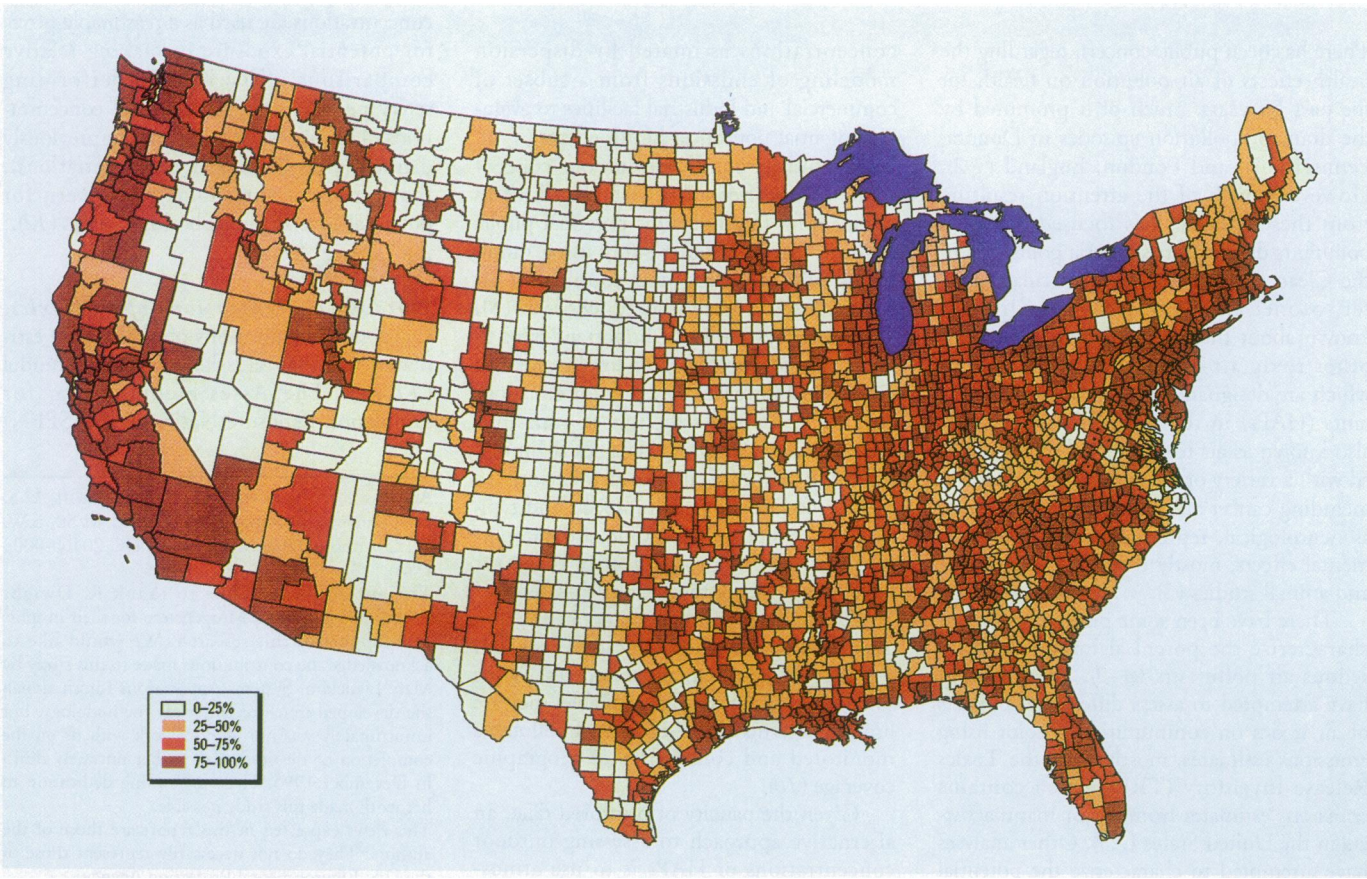

Figure 1. Distribution of total modeled air toxic concentrations by county in the contiguous United States in 1990, shown in quartiles.

national VOC and PM emissions inventories do not include data for these states.

The dispersion model accounted for long-term concentrations of HAPs attributable to current (i.e., 1990) anthropogenic emissions within 50 km of each census tract centroid. For 28 HAPs, estimated outdoor concentrations also included a background component attributable to long-range transport, resuspension of historical emissions, and natural sources derived from measurements taken at clean air locations remote from the impact of local anthropogenic sources (*11*).

Cancer and noncancer benchmark concentrations. Benchmark concentrations were derived from available toxicity data on carcinogenic and short- and long-term noncarcinogenic effects for each HAP (*12*). A benchmark concentration represents a level of potential regulatory and public health concern. Concentrations posing a one-in-a-million cancer risk were used as benchmark concentrations for cancer effects. The EPA's inhalation reference concentrations (RfC), or similar values developed by other agencies, representing levels below which long-term exposure is not expected to result in any adverse health effects, were selected as the benchmark concentrations for noncancer health effects from long-term exposure. Levels of Concern (LOC), established for chemicals on the list of extremely hazardous substances in the Superfund Amendments and Reauthorization Act, section 302 (*17*), were divided by a safety factor of 1,000 (LOC/1,000) and chosen as screening indicators of potential noncancer hazards from short-term exposures to HAPs. The factor of 1,000 was suggested as an appropriate crude estimate of the factor needed to convert the LOC, based upon mortality or very serious effects, into a level which would ensure that no adverse health effects would be observed and to address concerns about the uncertainty of the estimate (*3*). Toxicity data were compiled from various regulatory sources including the EPA, the California Environmental Protection Agency (Cal-EPA), and the Agency for Toxic Substances and Disease Registry (ATSDR) (*12*).

Fourteen of the 148 HAPs included in this study are chemical groups. For example, the HAP listed as mercury compounds is made up of several different constituents, including mercuric chloride, elemental mercury, mercuric nitrate, and mercury (aceto) phenyl, all with potentially different levels of toxicity. Thus, it is difficult to assess the toxicity of chemical groups because each is comprised of a number of different constituents that may have varying levels of toxicity. For this paper, toxicity values that can be assigned to an entire chemical group are included (*12*). Toxicity values applicable only to individual constituents of chemical groups are not included because the modeled concentrations developed in this study represent the entire group.

Toxicity values were separated into two tiers, with those values having the highest level of data quality, consistency in derivation, and peer review assigned to Tier I. Details on the rationale and methodology for prioritizing hazard data are discussed elsewhere (*12*). Table 1 shows the classification of toxicity data for deriving benchmark concentrations used in this paper.

Comparisons of estimated HAP concentrations to benchmark concentrations. The modeled concentrations developed in this study represent long-term outdoor concentrations present in any one location. To screen for whether a modeled concentration represents a potential health risk to the general population, it is compared to benchmark concentrations for cancer and noncancer effects. A HAP may have both cancer and noncancer benchmarks. A modeled long-term concentration greater than a cancer or chronic benchmark is considered to be an indicator of potential adverse health effects.

Estimated outdoor concentrations were also compared to benchmarks for health effect concerns from short-term exposure. While the estimated concentrations in this analysis do not represent short-term peak concentrations, exceedance of short-term benchmarks by annual average concentrations strongly suggests that transient peak concentrations may also be too high.

Comparison of estimated HAP concentrations to benchmark concentrations implicitly treats outdoor concentrations as equivalent to exposure concentrations. Outdoor concentrations are a reasonable proxy for exposures that occur both outdoors and indoors, given the high rates of penetration into indoor environments for various HAPs (*18,19*).

Hazard ratios were computed for each available benchmark for each census tract by dividing each estimated HAP concentration by the HAP's benchmark concentrations for both cancer and noncancer effects. Hazard ratios greater than 1 indicate that the estimated concentration exceeds the benchmark concentration.

Results

Figure 1 shows the distribution of total modeled HAP concentrations of each county, by quartile, across the contiguous United States. Total modeled HAP concentrations are the unweighted sum of the estimated concentrations of all 148 HAPs. The county level concentrations represent average concentrations of the census tracts in each county. Most of the top quartile of total HAP concentrations occur in the industrialized areas of the West and East Coasts, around the Great Lakes, and along the Gulf of Mexico. Other methods for weighting concentrations of HAPs result in similar spatial distributions. There can be considerable variability in the concentrations among the census tracts within a county. The coefficient of variation of the total HAP concentration for the census tracts within each of the counties ranged from 0 to 180%, with a median value of 21%.

Comparison of estimated HAP concentrations in each of the census tracts to benchmark concentrations showed that there were a number of benchmark concentrations exceeded in a majority of the census tracts. Eight pollutants [benzene, carbon tetrachloride, chloroform, ethylene dibromide, ethylene dichloride, formaldehyde, methyl chloride, and bis(2-ethylhexyl) phthalate] had modeled concentrations exceeding the benchmark concentrations for cancer in 100% of the census tracts. For each of these HAPs, the background concentration alone, as defined above, exceeded the benchmark concentration for cancer, as shown in Table 2. To evaluate the impact of current anthropogenic emissions, the

Table 2. Background concentrations for hazardous air pollutants (HAPs) that exceed benchmark concentrations in all census tracts

Hazardous air pollutant	Background concentration ($\mu g/m^3$)	Cancer benchmark concentration ($\mu g/m^3$)[a]	Ratio of background to benchmark concentration	No. of census tracts with exceedances, disregarding background (%)
Bis(2-ethylhexyl) phthalate	1.6	0.25	6.4	18 (<1)
Benzene	0.48	0.12	4.0	56,000 (92)
Carbon tetrachloride	0.88	0.067	13	1,600 (3)
Chloroform	0.083	0.043	1.9	4,900 (8)
Ethylene dibromide	0.0077	0.0045	1.7	900 (1)
Ethylene dichloride	0.061	0.038	1.6	13,000 (21)
Formaldehyde	0.25	0.077	3.2	57,000 (94)
Methyl chloride	1.2	0.56	2.2	110 (<1)

[a]All cancer benchmarks are for Tier I carcinogens except for bis(2-ethylhexyl) phthalate.

background concentration was subtracted from the total estimated concentrations, with remaining concentrations compared to benchmark concentrations. Modeled concentrations for two pollutants (benzene and formaldehyde) exceeded the benchmark concentration in over 90% of the census tracts and one HAP (ethylene dichloride) exceeded the benchmark concentration in 21% of the census tracts (Table 2). The remaining five HAPs had concentrations from modeling of current anthropogenic sources that exceeded benchmark concentrations in a much smaller percentage of the census tracts.

Figure 2 shows the distribution of the number of benchmark concentrations exceeded by estimated outdoor concentrations per census tract across the United States. The lowest number of benchmarks exceeded in an individual census tract was 8 and the highest was 32, with a mean of 14. Approximately 50% of the census tracts, or 30,000, had between 11 and 15 estimated HAP concentrations that exceeded benchmark concentrations.

There was a wide range of hazard ratios for the HAPs. Figure 3 shows a quartile plot of the hazard ratios for cancer benchmarks classified as Tier I. Of these 40 pollutants, 35 had at least one census tract with an estimated concentration over the benchmark concentration. Thirteen HAPs had estimated concentrations at least 100 times the cancer Tier I benchmark concentrations (i.e., estimated concentrations greater than a 1 in 10,000 cancer risk level); such concentrations occurred in about 10% of the census tracts with total residential population of approximately 20 million people. Five HAPs did not exceed a benchmark concentration in any census tract and had a hazard ratio less than 0.01 in most census tracts. An additional 21 pollutants with Tier II cancer benchmarks had portions of their hazard ratio distributions above one, while three of these pollutants, lead, *p*-dichlorobenzene, and quinoline, had portions of their distributions above 100.

Figure 4 shows the quartile plot of the hazard ratios for pollutants with Tier I chronic toxicity benchmarks. Eight of these pollutants had some portion of their hazard ratio distributions above 1, with exceedances of the benchmark concentrations occurring in approximately 56,000 census tracts. One of these pollutants—acrolein—had a median hazard ratio greater than 1 and a maximum ratio of about 1,000. There were also approximately 200 census tracts with a residential population of approximately 220,000 that had hazard ratios for any pollutant with a Tier I chronic toxicity benchmark greater than 100. Fourteen additional

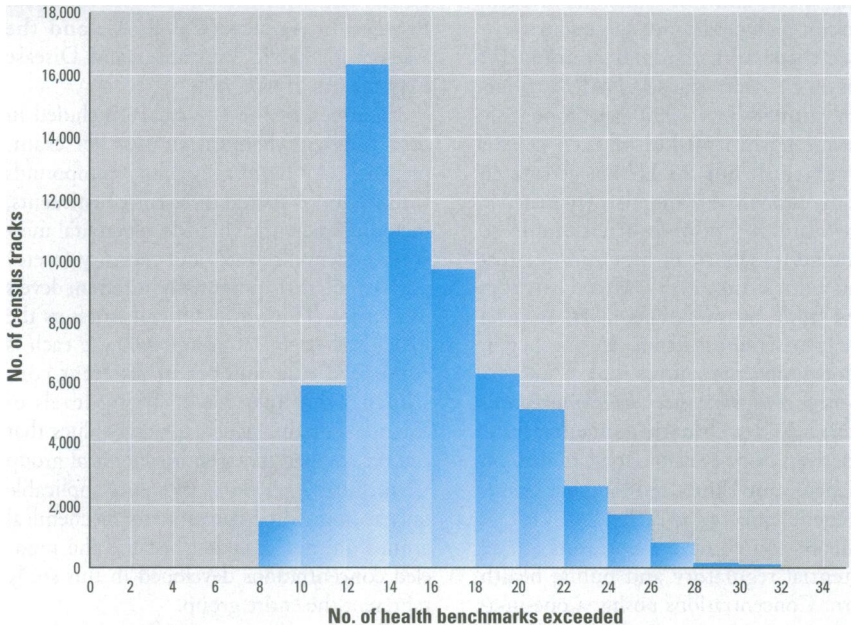

Figure 2. Distribution of the reestimated number of hazardous air pollutant benchmark concentration exceedances per census tract in 1990, considering all Tier I and Tier II benchmark concentrations.

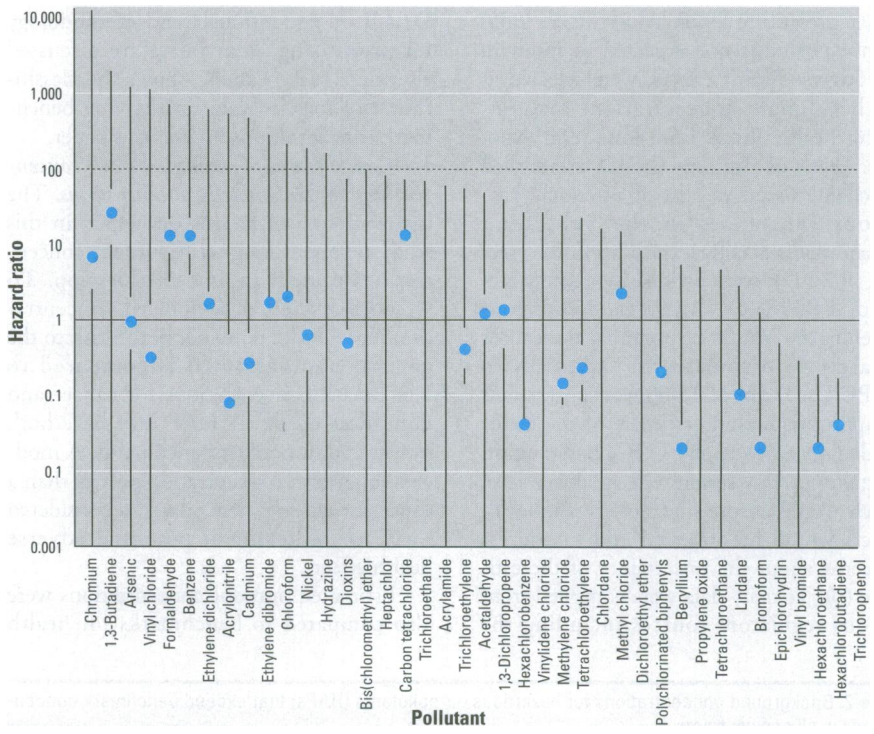

Figure 3. Quartile chart of the 1990 hazard ratios (defined as the estimated outdoor concentration divided by the benchmark concentration) for cancer benchmark values in Tier I. The lower line represents the 0–25th percentile, the dot the median value, and the upper line the 75th–100th percentile.

pollutants with Tier II chronic toxicity benchmarks had hazard ratios greater than 1. Of these, chromium had estimated concentrations at least 100 times the chronic toxicity benchmark in 12 census tracts, while the other 13 HAPs had maximum hazard ratios less than 15. The remaining 43 pollutants with chronic toxicity Tier II benchmarks had all hazard ratios below 1. In addition, 4 pollutants with acute toxicity benchmarks had hazard ratios greater than 1, with a maximum ratio of 40. Approximately 800 census tracts had at least one exceedance of an acute toxicity benchmark.

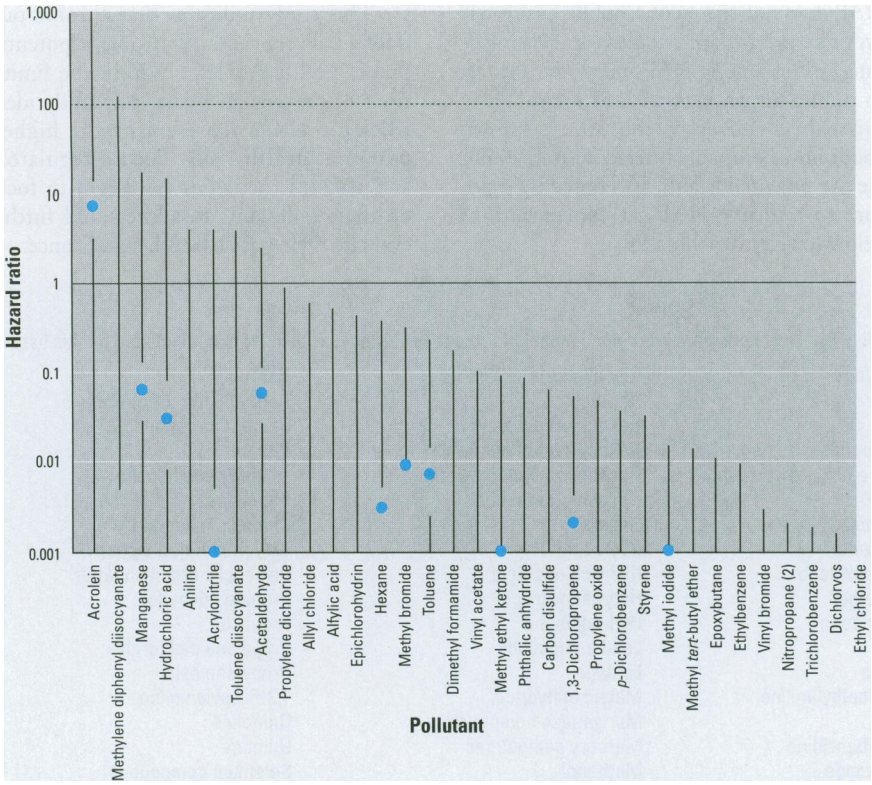

Figure 4. Quartile chart of the 1990 hazard ratios (defined as the estimated outdoor concentration divided by the benchmark concentration) for chronic toxicity benchmark values in Tier I. The lower line represents the 0–25th percentile, the dot the median value, and the upper line the 75th–100th percentile.

Discussion

This analysis of air toxics concentrations across the United States detected several HAPs that are ubiquitously high in comparison to benchmark concentrations. For some of these air toxics, the concentration was dominated by background concentrations made up of a combination of long-range transport, resuspension of historical emissions, and nonanthropogenic sources. For the HAPs with background concentrations that exceeded benchmark concentrations, carbon tetrachloride and ethylene dichloride have no known natural contributions to background, while the other six have some portion of background from both anthropogenic and nonanthropogenic sources (*20–22*). The relatively high background concentrations indicate a ubiquitous presence of these pollutants to which current and future emissions are added. If background is disregarded, two of these eight pollutants have modeled concentrations greater than the benchmark concentration in over 90% of the census tracts (benzene and formaldehyde).

This analysis uses emissions data for 1990, and changes in air toxics concentrations may have occurred since then. The EPA estimates that total emissions of VOCs, which include many of the HAPs in this analysis, decreased approximately 3% from 1990 to 1995 (*23*). Overall emissions of the VOC HAPs in this analysis may have experienced a similar decline during this time; however, changes in emissions and concentrations of individual HAPs will probably vary substantially because the decline in VOC emissions was not constant across source categories. Estimated on-road mobile source emissions of VOCs declined by about 11% from 1990 to 1995; corresponding reductions in mobile source emissions of HAPs such as benzene, 1,3-butadiene, and formaldehyde may be likely. Decreases in mobile source emissions may be offset to some extent by increases in VOCs emitted by several other source categories because the EPA estimates indicate increases of VOC emissions for chemical and allied product manufacturing (6% increase), other industrial processes (5%), solvent utilization (7%), waste disposal and recycling (7%), and nonroad mobile sources (6%) from 1990 to 1995 (*23*).

The results from this analysis are limited by incomplete hazard data. Most of the hazard ratios greater than 1 were for cancer benchmark concentrations. This is consistent with the presence of a large number of carcinogenic HAPs in section 112 of the Clean Air Act and the conservative one-in-a-million benchmark used. However, incomplete toxicity information for the HAPs must be considered when assessing potential health impacts. Approximately 20% of the modeled HAPs with a weight of evidence indicating potential carcinogenicity do not have a cancer value, and 50% do not have a benchmark concentration for noncancer health effects (*12*). Even for some of the ubiquitous pollutants identified in this analysis, there is incomplete toxicity information. For example, benzene and 1,3-butadiene have both been associated with reproductive and developmental effects (*3*), but they currently have no benchmark concentrations for such effects. Finally, 29 of the 148 HAPs included in this study have no Tier I or Tier II benchmark concentrations for any effects, even though there are previous studies indicating some of these HAPs are of potential health concern (*3*). For example, *N,N*-dimethylaniline has been characterized by the EPA as being of high concern for noncancer effects, but quantitative hazard information is not available (*3*).

Another limitation in the toxicity information for the HAPs is in hazard evaluation for chemical groups. Outdoor concentrations of HAPs were estimated for 14 chemical groups. It is difficult to assess the toxicity of chemical groups because they are composed of a number of different species. For example, the HAP listed as mercury compounds is made up of several different constituents including mercuric chloride, elemental mercury, mercuric nitrate, and mercury (aceto) phenyl, all with potentially different levels of toxicity. Future work will evaluate the constituents of the chemical groups and their potential toxicity. The incomplete assessment of the toxicity of the HAPs, including both unquantified effects and incomplete information on chemical groups, limits the ability to fully assess the potential health significance of the modeled HAP concentrations.

The modeled concentration estimates developed in this study have a general tendency to underestimate HAP concentrations found by the limited monitoring data available (*11*). This could result in underestimates of the frequency with which benchmark concentrations were exceeded in 1990. In addition, the modeled concentrations do not capture spatial or temporal peak concentrations that could be significant. The available monitoring data support this study's conclusion that exceedances of benchmark concentrations are common. For example, several sources of long-term monitoring data for benzene and 1,3-butadiene show that measured concentrations routinely exceed benchmark concentrations (*11*).

This analysis only considers the potential health impact of individual pollutants,

though the results indicate a concern for multiple pollutants in combination. Census tracts across the United States were predicted to have a mean of 14 HAP concentrations greater than the benchmark concentration, indicating simultaneous high concentrations of multiple HAPs. An estimated HAP concentration less than the benchmark concentration may indicate that the HAP does not represent a public health risk on its own. However, additive or synergistic interactions among HAPs may pose a threat to public health beyond that identified in this paper. Currently, too little is known about how pollutants interact to fully evaluate the potential health risks posed by exposure to multiple HAPs at concentrations below toxicity benchmarks.

This study indicates that chronic outdoor HAP concentrations pose a potential public health problem. Within the limitations of the available data, this study identifies the HAPs representing the highest potential health risks. Future regulatory and scientific activities can begin to focus on these pollutants to address and further evaluate their public health significance.

Appendix

List of 148 hazardous air pollutants included in this study (1990 emissions data were not available for those hazardous air pollutants not included on this list).

Acetaldehyde
Acetamide
Acetonitrile
Acetophenone
Acrolein
Acrylamide
Acrylic acid
Acrylonitrile
Allyl chloride
Aniline
Anisidine
Antimony compounds
Arsenic compounds
Benzene
Benzotrichloride
Benzyl chloride
Beryllium compounds
Biphenyl
Bis(2-ethylhexyl) phthalate
Bis(chloromethyl) ether
Bromoform
1,3-Butadiene
Cadmium compounds
Calcium cyanamide
Captan
Carbaryl
Carbon disulfide
Carbon tetrachloride
Carbonyl sulfide
Catechol
Chloramben
Chlordane
Chloroacetic acid
Chlorobenzene
Chloroform
Chloromethyl methyl ether
Chloroprene
Chromium compounds
Cobalt compounds
Cresol
Cumene
Cyanide compounds
2,4-Dichlorophenoxyacetic acid salts and esters
Dibutylphthalate
p-Dichlorobenzene
3,3′-Dichlorobenzidene
Dichloroethyl ether
1,3-Dichloropropene
Dichlorvos
Diethanolamine
N,N-Diethyl/dimethylaniline
Diethyl sulfate
3,3′-Dimethoxybenzidine
Dimethyl formamide
1,1-Dimethyl hydrazine
Dimethyl phthalate
Dimethyl sulfate
4,6-Dinitro-*o*-cresol
2,4-Dinitrophenol
2,4-Dinitrotoluene
1,4-Dioxane
Dioxins/furans (toxicity equivalents)
Epichlorohydrin
1,2-Epoxybutane
Ethyl acrylate
Ethylbenzene
Ethyl carbamate
Ethyl chloride
Ethylene dibromide
Ethylene dichloride
Ethylene glycol
Ethylene oxide
Ethylene thiourea
Ethylidene dichloride
Formaldehyde
Glycol ethers
Heptachlor
Hexachlorobenzene
Hexachlorobutadiene
Hexachlorocyclopentadiene
Hexachloroethane
Hexane
Hydrazine
Hydrochloric acid
Hydrofluoric acid
Hydroquinone
Lead compounds
Lindane
Maleic anhydride
Manganese compounds
Mercury compounds
Methanol
Methoxychlor
Methyl bromide
Methyl chloride
Methyl chloroform
Methyl ethyl ketone
Methyl hydrazine
Methyl iodide
Methyl isobutyl ketone
Methyl isocyanate
Methyl methacrylate
Methyl *tert*-butyl ether
4,4′-Methylene bis(2-chloroaniline)
Methylene chloride
Methylene diphenyl diisocyanate
4,4′-Methylenedianiline
Naphthalene
Nickel compounds
Nitrobenzene
4-Nitrophenol
2-Nitropropane
Parathion
Pentachloronitrobenzene
Pentachlorophenol
Phenol
p-Phenylenediamine
Phosgene
Phthalic anhydride
Polychlorinated biphenyls
Polycyclic organic matter
Propionaldehyde
Propoxur
Propylene dichloride
Propylene oxide
1,2-Propylenimine
Quinoline
Quinone
Selenium compounds
Styrene
Styrene oxide
1,1,2,2-Tetrachloroethane
Tetrachloroethylene
Toluene
2,4-Toluene diamine
2,4-Toluene diisocyanate
o-Toluidine
1,2,4-Trichlorobenzene
1,1,2-Trichloroethane
Trichloroethylene
2,4,6-Trichlorophenol
Trifluralin
2,2,4-Trimethylpentane
Vinyl acetate
Vinyl bromide
Vinyl chloride
Vinylidene chloride
Xylene

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